and the relative amount of difference in time so spent continues to increase from that time onward. It cannot be urged that the period of puberty in girls has an effect in causing this difference, since the difference begins at a much earlier time of life and reaches its climax before that age in females. The delay in the onset of this increase in females in England and the lessened difference at the age 10-15 years may, perhaps, be accounted for by climatic differences, that is, a generally milder climate in the British islands.

A singular instance of the hold which popular fallacy in regard to infection gains upon the laity is presented in the results of a legislative inquiry which occurred in Massachusetts last winter, but which, fortunately, proved a failure.⁶

The underlying principle which could have any possible weight in this investigation was the possible danger of the spread of tuberculosis infection from a hospital inhabited by consumptives to the surrounding neighborhood, or from its inmates while walking about the grounds or the neighboring streets; or, in other words, the danger of outdoor infection. Since such danger was neither proven, nor was even a strong probability thereof shown to exist, the proposed measure utterly failed.

Once let the idea of infection gain a foothold in the popular mind, and the belief spreads rapidly and often to such an extent as to cause harm instead of good. The method and degree of infection are all of no account so long as these words "infectious or contagious" have become impressed upon the mind of the community.

Modern medicine has received no greater impetus than that which has been derived from the rapid advances afforded by bacteriological research. So great, and so valuable have these results proved, that the death-rate from some of the worst forms of infection has begun to lessen with greater rapidity than before, under the influence of methods which had their origin in the laboratory of the scientist.

Notwithstanding this fact, however, it cannot be denied that the most reliable conclusions in regard to the true characteristics of infectious diseases must be derived not only from the laboratory but from the observations of the physician in his daily rounds of practice, together with a study of mankind in his relation to all outward circumstances which influence his condition in life, such as those of sex, age and occupation. In other words, the results of laboratory investigation, clinical observation, and the broad and general study of the natural history of disease taken together constitute a better means of deciding doubtful questions in regard to infection than either of these methods taken separately.

In conclusion, then, it must be admitted, that tuberculosis is an infectious disease. Its character is too well defined, both by observation and by experiment, to admit a reasonable doubt. The active principle of infection, the bacillus of tuberculosis is recognizable; it is capable of isolation from the body, of cultivation, and of inoculation into the lower animals. But, practically, the exact mode of infection, when applied to the daily intercourse of human beings, is extremely limited, and no evidence has ever been presented which tends to show that such infection ever occurs by exposure in the open air, either by the breath of con-

sumptives or by the distribution of sputum upon the surface of the soil. On the other hand, abundant evidence shows that such infection is of very common occurrence from indoor exposure to the distribution of sputum deposited upon floors and dried under favorable circumstances.

By this I do not mean to infer that the outdoor free distribution of the sputum of consumptives should be encouraged or allowed, but should be prevented, on the general principle that all infectious material constitutes a greater or less danger, and not because any serious danger from such outdoor distribution has actually been shown to exist when compared with indoor distribution of sputum.

SOME CONSIDERATIONS OF TUBERCULOSIS.¹

BY J. ARTHUR GAGE, A.M., M.D., LOWELL, MASS.

FREQUENCY.

TWO-SEVENTHS, or nearly 80 per cent., of civilized people are said to have tuberculosis. If this is true, then Lowell has 26,000 people afflicted with tuberculosis. The figures seem appalling. What is the evidence? In 1,000 autopsies by Biggs of New York, 275 or 27.5 per cent. had lesions of tuberculosis. If we consult our city mortality tables we find that for the last thirty years between 15 per cent. and 20 per cent. of all deaths are from the tuberculoses, and if we consider the cases of tuberculosis not only of the lungs, but of bones, skin, glands and other organs that we see maintaining a degree of health consistent with life, these figures do not seem extravagant. But if we look at the mortality charts we notice a curious fact. In 1884, the curve representing the per cent. of deaths from tuberculosis to the total deaths went for the first time below 15 per cent. and has steadily declined since, until it has reached 10 per cent. In the same year the curve representing a corresponding per cent. of deaths of children under five years began steadily to rise from 36 per cent. and has gradually reached a level of 43 per cent. In general this curve is influenced by the deaths from zymotic and diarrheal diseases (the causes of death in most children), but if we take the year 1892, when there were few deaths from all zymotic diseases and when the deaths from diarrheal diseases were the average for ten years, that is when we should expect a low death-rate among children, we find that 42 per cent., nearly the highest for ten years, of deaths were under five years. The deaths from diarrheal diseases was 12 per cent., and from zymotics 21 per cent., making a total of 33 per cent. So that we have 9 per cent. of the total deaths among children under five years to account for. What are the causes given for these deaths?

If we look at the last bulletin, we find that of 83 deaths under five years, 19 are classified as infantile debility and inanition. (Of course the responsibility for this does not rest upon the Board of Health.) In other words, a great many deaths among children really have no pathological cause of death given. Yet we know that during the first two years of life there is a constant increase in tuberculous disease, which then falls only to rise again at puberty.

To recapitulate: When we consider that the death-

⁶ The circumstances attending this investigation, having been already presented in the Journal, it is unnecessary to repeat them here.

¹ Read before North Middlesex District Medical Society, October 27, 1897.

rate under five years of age is higher than the average for thirty years, and out of proportion to the death-rate from zymotic and diarrheal diseases; that the death-rate from tuberculosis is lower than ever before; and that there are many unclassified deaths at an age when we know tuberculosis to be rife; I believe the inference is warranted that many of these are due to tuberculosis, and that the death-rate from that disease should be higher than it is.

Since I began private practice I have been impressed with the great frequency with which signs of solidification have appeared in some portion of the lungs in young people under twenty years of age under varying conditions, and these conditions have often seemed inconsistent with the supposition that, at the particular time when the signs were noticed, the patient was infected with tubercle bacilli. A few cases will illustrate:

(1) Boy, fourteen, doubtful family history, convalescing from typhoid, has suddenly appear all the signs of solidification at right apex, with fine and coarse moist râles. With forced feeding the râles disappear, and the boy resumes good health.

(2) Sister of above, eighteen years old, after over-work catches cold, and has same signs at right apex; recovers health, but never gets back to former weight.

(3) Girl, thirteen years old, has a cold, with cough and localized signs at right apex. Symptoms subside under treatment, and girl recovers health and weight.

It is interesting to note that the girl of eighteen does not regain weight, while the girl of thirteen does, and also that no one of the three lived with people in active consumption.

(4) A girl of seventeen "feels tired," and does not want to go to school or work; one brother died at twenty-one of phthisis. The father and mother are healthy, but have a tubercular family history. No signs of active disease exist in the lungs, but there are prolonged expiratory murmur and increased voice at right apex. Later, active symptoms develop. One would naturally think of the connection with the brother's case; but the development was two years later, and it is significant that another brother seven years old had "lung fever" years before either was sick. I frequently see the signs of local disease of lungs in young children under two years, but will return to this subject later.

It is not necessary to multiply cases, but I want to call attention to the frequency of localized inflammations of the lung in children, particularly about puberty. These patients, so far as we can see, are not associated with active consumption in their daily life, nor can they be said to be especially exposed at the time their symptoms appear. It is evident that many of these cases will be seen with lung trouble later in life, and the attack then will be the lighting up of the old lesion; such is likely to happen about the age of twenty-one or twenty-two; and the query comes, If these cases carried the bacillus in their bodies from thirteen years to twenty-one, when did they get it? May they not have had it in the earliest period of life, under two years, when we know so many children have tuberculosis? If they did acquire the disease in a latent form at so early a stage, how did they get it, and what are the sources of contagion?

METHODS OF INFECTION.

Over two years ago, in a discussion before the Massachusetts Association of Boards of Health in Boston, I

reported some cases of inflammation of the lung, probably tubercular, occurring in children, in the early months of life when the child seemed to be free from danger of infection, that suggested to my mind the possibility of the inheritance of the bacillus. This suggestion was vigorously opposed, and this opposition has served as a stimulus to seek further evidence. Let us look at a few cases:

CASE I. A boy is born to parents neither of whom have had active tuberculosis. One family (mother's) is tubercular, and the mother herself had suspicious signs at right apex of some solidification, but she does not cough or expectorate. The baby is fed on sterile milk from birth. The house is new; no consumptive lives in house. At four months of age he has an inflammation of posterior lobe of one lung that lasts several weeks and leaves him very weak; no sputum examination could be made. For two years he is pale and under weight; has "wheezy" breathing and some cough, and moist râles can be heard at times over the old spot of inflammation. Slowly he builds up on cod-liver oil and fresh air.

CASE II. A boy is born to parents both of whom are in vigorous health, full weight, and free from any history of tuberculous disease. The mother's lungs are without even suspicious signs. Father's family is tubercular. The baby is nursed by mother. No consumptives have lived in house, so far as known, in recent years. At six months of age the boy begins to lose flesh and strength, and become pale: there are no other symptoms. His digestion is good, and he is given juice of beef and tonics, with no improvement. At nine months of age a consultation is held; and after thorough physical examination it is agreed that there are signs of solidification of one base. Under forced feeding with Pasteurized milk, beef-juice and cod-liver oil (nursing is stopped) he gains. The stomach digests all he gets, but he still is very pale.

These taken as two sample cases, with many other cases of tuberculosis of lungs, meninges and bones in the first year of life, bring us to the question, Can it be that these cases inherit the bacillus? What is the clinical evidence? If we glance at the literature of the subject, we shall find that since the time of Hippocrates physicians have believed that people inherited a "consumptive tendency"; and I doubt not but that all of you to-day question a patient with pulmonary symptoms about his family history, not merely of those he lives with, but as to his grandparents, uncles and aunts, and collateral branches. Now, under this name "tendency" we have merely covered up our ignorance. For whether we interpreted it to mean lowered vitality or decreased nutrition or a mysterious property called "susceptibility," we were merely trying to explain the fact that "consumption seems to run in families." Do not understand me as saying that a child of a consumptive may not be less resistant to acquired tubercle bacilli than children of healthy parents; but it may be fairly questioned why they should be more susceptible than children of parents with other debilitating diseases. How, then, shall we explain this "susceptibility"? Can we say with Cohnheim, that "the phthisical habit is not an indication of a tendency to, but actually of the existence of tuberculosis"?

The question divides itself into three:

(1) Can the bacillus be transmitted to the ovum by the mother or the sperm of father?

(2) To fetus by mother?

(3) How often does this occur?

In answer to the first two questions, we know that micro-organisms can be transmitted from mother to child in utero. How this is done through the placenta is foreign to our present discussion. In the case of syphilis (presumably a germ disease) we know that it can be transmitted through the ovum and sperm direct, thus coming from both parents. But it has been shown (quoted from Dr. W. H. Welch, of Johns Hopkins) that the bacilli of chicken cholera, anthrax and pyogenic micrococci pass through the placenta of animals. In man we know that the fetus in utero has been infected with small-pox, measles, scarlet fever, relapsing fever, typhoid, anthrax and other diseases.

In regard to tuberculosis, Welch has the following to say: "Developed tuberculosis in the new-born infant is extremely rare, although it has been observed, but this rarity does not prove that tubercle bacilli may not often be transmitted from mother to fetus. It takes time and susceptibility for tubercle bacilli to produce recognizable lesions. Tubercle bacilli without developed tuberculosis have been repeatedly found in the fetus of tuberculous mothers. Birch-Hirschfeld detected in a seven-months' fetus, removed by Cæsarean section from a mother with acute miliary tuberculosis, tubercle bacilli, both by microscopical examination and by inoculation of guinea-pigs. There were no tuberculous lesions in the fetus. The failure to find tubercle bacilli in a large number of similar cases can hardly be regarded as proof of their absence, when one considers the difficulty of demonstrating tubercle bacilli in small numbers irregularly distributed through a large mass, without any lesion to indicate where they are most likely to be found. Gartner finds that the transmission of tubercle bacilli from mother to fetus is common in the experimental tuberculosis of mice, rabbits and canary birds.

"There is reason to believe that the embryo offers great resistance to the growth of tubercle bacilli. Maffucci has found that tubercle bacilli inoculated into hen's eggs before incubation remained quiescent during the period of embryonic development, but caused the death of most of the chicks from tuberculosis in three weeks to four and a half months after birth. We have evidence that in human beings living and virulent tubercle bacilli may remain latent in the body a long time. As already mentioned, the chances of penetration of bacilli into the fetus appear to be more favorable during the later period of pregnancy than earlier. The frequency of tuberculosis increases very rapidly with each succeeding week after birth, until during the second half of the first year and during the second year of life fatal tuberculosis is very common, and then becomes less frequent until after puberty. Infantile tuberculosis is far more commonly situated in internal organs, such as the lymphatic glands, meninges, bones and joints, without tuberculous lesion on any exposed surface of the body, than is tuberculosis of adults."

Thus it is pretty conclusively shown that the mother can transmit the bacillus to offspring. What about the father? Osler says, "There is no evidence to show that a tuberculous father can transmit the disease." Let us consider this question further. In syphilis we know that the semen can transmit a constitutional disease to the offspring. Again, we know

² Dennis's Surgery, vol. 1, p. 307.

that the human semen contains under certain conditions the tubercle bacilli. Now, let us turn for a moment to animals for additional light.

In a most valuable and interesting paper by Dr. G. H. Bailey, State Veterinarian of Maine (which I wish I had time to quote fully) the following facts are brought out: 14 bull calves a few days old were sold from an infected herd to go to different parts of the State. Twelve of the 14 were later found diseased, and their calves out of sound cows were also found diseased. He believes they were born with the disease, and transmitted it directly to their offspring, as there had never previously been any tuberculosis on the farms to which they carried the disease.

A second case was that of a breeder who bought an infected bull and served 10 cows, all the calves of these 10 cows had to be killed for tuberculosis.

In a third case where an infected bull served seven cows, all their calves were tuberculous, and one tuberculous calf came from a mother free from the disease. Hence, the disease *could* be traced directly from the bull.

He quotes as follows:

Dr. Theobald Smith of Massachusetts State Board of Health says: "(1) The tubercle bacillus may pass in the semen of the male and infect the ovum directly. (2) The ovum may be infected by disease of surrounding structures (peritoneum, ovaries, Fallopian tubes) in the female. (3) The fetus may be infected by the passage of tubercle bacilli from the maternal placenta into the fetal circulation. The infection through the placenta is probably the most frequent cause of congenital tuberculosis. It may take place when, in consequence of generalized tuberculosis of the mother, tubercle bacilli are in the circulation and become lodged in the placenta, whence through some ruptured vessel, they may pass into the blood of the fetus."

From *Oest Monatschrift* he quotes as follows: "The presence of microbes in the spermatid fluid, however, is of far greater importance, as it throws a new light upon the transfer of the microbe from father to child, not only in tuberculosis but in syphilis."

From Dr. Michenor, Chief Inspector of the Bureau of Animal Industry, Washington: "This infected bull is an unsafe sire, and is almost certain to transmit the disease."

The Maine State Board of Health says: "Tuberculosis in cattle is a hereditary disease."

The Brussels Congress, 1888, says: "The disease (tuberculosis) is transmitted by heredity."

The New York State Commission on Tuberculosis in cattle says: "Tuberculosis is under certain conditions congenital."

Many other quotations might be given; but enough has been heard to show that our friends the veterinarians believe that the disease is *frequently* transmitted from father to offspring.

Believing, as I do, in the universality of law, I think that if a bull can transmit tuberculosis to his offspring, a man can do the same thing.

We now come to the question, How often does this happen in man? And I wish to say here, that I believe the answer to this question must rest upon most careful work and observation in the future, but that its importance cannot be overestimated. We can only at present consider some of the evidence that favors it. On the part of the child the fact that

so many children have tuberculosis during the first two years of life would point to inheritance, where other sources of contagion have been eliminated; but if it can be shown that often tuberculosis in children is latent and is really the focus of later outbreaks, then I should consider this to point rather towards inheritance, because I should expect acquired tuberculosis to develop a lesion rather than to remain latent.

This tendency of acquired tuberculosis to develop a lesion was well illustrated in case of a young man of good family history and superb physique who, after engagement to a woman with apex disease, showed during an attack of persistent dry cough a spot about the size of a dollar in *supra spinous fossa* that gave all the classical signs of tuberculosis.

In a paper by Dr. Donaldson, President of the American Climatological Association, read in 1887, he says: "The greatest objection to the theory of inherited bacilli is that it is necessary to assume a latency of the disease during early life."

But we all know that the disease has the property of quiescence during all periods of life and for varying lengths of time, and I cannot see that latency means anything more than the property of not producing evident lesions, or giving rise to active symptoms. When we see a child with tuberculosis at one year maintaining good health for years, or a boy at fourteen years with undoubted tuberculosis growing up without active disease, I call that latency. The experiment of injecting tubercle bacilli into chicken's eggs and not getting the disease (manifested by tangible lesions) until three to fourteen weeks of age is strong proof of latency; while in cattle, the evidence is all in favor of the latency of tuberculosis.

Dr. F. W. White's experiments with tuberculin bear upon this point. About 20 per cent. of cases injected with tuberculin, where no physical evidence of tuberculosis could be found, reacted. This was noted as a failure of tuberculin. When we consider the frequency of tuberculosis and the fact that no such discrepancy is found in cattle, it seems to me fully as sound to assume that that per cent. of patients had latent tuberculosis that presented no symptoms discoverable on physical examination.

Furthermore, the lesions of tuberculosis found at autopsy, that were quiescent, all point the same way. Consequently, if the difficulty of explaining latency in tuberculosis is the great objection to the theory of frequent inheritance of tuberculosis, then that objection seems to me to be a slight one.

If we look at the question from the standpoint of the parents, it seems probable that a mother with tuberculosis of the ovary, or a father with bacilli in sperm, would be in a favorable condition to transmit the disease; or a woman with multiple lesions of tuberculosis over the body, and the bacilli carried in the blood current, if she should become pregnant, would be very likely to transmit the bacilli, particularly if there were any break in the placental membrane.

In conclusion, it seems to me safe to say that the transmission of bacilli from parent to offspring is (upon the evidence) much more frequent than has been believed. Can we say that predisposition means bacilli in the body? I cannot better reply than by quoting Ruhle before German Congress, 1887:

"If the inheritance of human consumption be true, and we believe it; if the bacilli represent the infectious

material of tuberculosis, and we entertain no doubt of this, then both facts must harmonize with each other. . . . Whether it is the tubercle parasite itself which is transmitted, or a certain constitution of nutritive substratum on which the universally present germ may lodge, we can only know by further investigation."

INFECTION BY MILK.

Another mode of infection that has been underestimated is through milk, both bovine and human. The following cases furnish evidence on that point.

CASE I. This case has been reported in print, but I will repeat briefly. Baby under one year. Family history free from tuberculosis. Parents and older sister in perfect health. House new. Baby nursed to nine months, and then was fed on one cow's milk. Child died of tubercular meningitis. Cow was examined and appeared healthy. Cow's milk injected into guinea-pigs: killed in six to eight weeks with typical tuberculosis. No disease of udder could be found.

CASE II. Girl, eighteen years old, good family history; came for general tuberculosis of glands of neck. She had had the disease about a year. She had drank milk regularly. During the year one of the family cows was killed for tuberculosis.

CASE III. A man of thirty-two; good family history; always healthy; wife and children healthy; living in a new house. In September had an attack of renal colic. After recovery he went to work on a farm for three weeks, and during that time drank a large quantity of milk daily, then returned to his work as motorman. At this time he noticed a small nodule, like a pea, under the right jaw, which gradually grew larger; and in the spring I removed the right submaxillary gland and some lymphatics that were tuberculous. In the intervening December a cow was killed for tuberculosis on the farm where he drank the milk.

CASE IV. A young man of twenty-one, family history good (sister looks tuberculous), was seen with upper half of left lung showing signs of old tuberculous disease and the lower half showing signs of recent invasion. Temperature 103°; pulse 120, with night-sweats; hemorrhage and loss of flesh; abundant tubercle bacilli in sputum. I ordered seven hours a day of sunlight and rest in easy chair on piazza, Pasteurized milk, and little morphia-sulphate for cough. In one week the temperature became normal in the morning and 100° at night, and he gained one and a half pounds; cough improved, and night-sweats nearly disappeared. He had had cough for a long time, but became worse this summer in spite of a vacation, when he gained in weight. His sister had a young baby during the summer, and the family milk-supply was changed for one cow's milk, which made the baby "so sick that they had to stop feeding it on the milk." He usually drank a glass before going to bed. Soon after the baby's sickness the cow was killed for tuberculosis.

I might give more such cases, and the literature of the subject is full of them. Probably 20 per cent. of the cows in Eastern Massachusetts are tuberculous, and about one-half of these have the bacilli in their milk, whether there is disease of the udders or not. Their milk furnishes an excellent nutritive medium to convey a virulent germ to the consumer, and the mixing of the milk but infects the whole quantity, while it dilutes the strength. This makes the danger from milk a real one of much greater magnitude than is generally recognized; and that the danger is not

merely for infants and invalids, but that healthy individuals can be infected is shown by the cases quoted.

In the case of children and typhoid patients, I believe the danger is very great, because they drink large quantities of milk with no guarantee of its purity, and are in a condition of feeble resistance. There is, of course, but one remedy at present, and that is, to keep all milk at a temperature of 165° F. for thirty minutes before using.

I will not drink raw milk myself, and I have the family supply Pasteurized daily. The danger from butter is a real one.

The danger from nursing a tuberculous mother is so well recognized and the remedy is so obvious that I will not consider this source further.

INFECTION BY AIR.

The third source of infection, namely, from the air, is so well recognized that I shall only speak of a few of its limitations. It seems to me that this mode of infection bears more than its share of responsibility at present. In spite of Cornet's statistics from hospitals and cloisters and the conclusions of the New York bacteriologists I cannot feel that efforts to free the air from bacilli are going to make such an amazing difference in the amount of tuberculosis. I am well aware that the bacillus has been found in outside air, and the air and dust of rooms; that it has been traced into the nostrils of human beings. But the sunlight and oxygen and dilution must minimize the danger outside, and until there is conclusive evidence that the large percentage of cases is due to breathing the infected air indoors to the exclusion of other sources of infection, I cannot feel that the drastic measures recommended for prevention are needed. This is particularly true for the large number of cases of tuberculosis that are not in an active stage.

One more mode of infection is worthy of consideration, because it seems to me its importance has been underestimated. This is direct contagion. Our surgical experience has certainly taught us the important part that direct contact plays in infection by other germs, and I cannot see why tubercle bacilli should not follow the same rule.

The methods by which tubercle bacilli can be transferred from one person to another are many and obvious and need not be gone into here. But I would like to call attention to the fact that bacilli are often carried from one person to another in a moist state; that the interval of time between the germ leaving one person and reaching another is shorter by this method, and that presumably this is a virulent form of infection.

DIAGNOSIS.

I wish to speak of the symptoms and signs of latent or quiescent pulmonary tuberculosis, because I believe that it is often overlooked. A patient comes complaining of varying symptoms, which in the composite give a picture generally of a loss of physical vigor. Symptoms referable to the lungs are usually absent, nor can any history of any previous lung disease be elicited. Cough is seldom complained of (in fact) often denied; but on cross-examination you can often get the statement that they habitually "clear the throat" on rising in the morning. One of the most marked cases I ever saw, one I have watched for twelve years, a man who denied all symptoms of disease of the lungs, finally

acknowledged that sometimes he cleared his throat in the morning. He managed to get just enough sputum to moisten the bottom of the bottle and it was found to be teeming with tubercle bacilli, which later completely disappeared. Hemoptysis is often absent in these cases. Low weight (that is, below average) is often present, though sometimes the patients are normal in this respect. Indigestion, a "tired feeling," and palpitation with some shortness of breath on exertion are occasional symptoms.

When we come to the physical signs we often find an incomplete picture.

On inspection we sometimes find slight depression above the clavicle, as compared with opposite side,—in one case of a strong man in good health and full weight, without pulmonary symptoms, a marble could rest in the depression above clavicle on the affected side, which in my experience is much oftener the right than the left. This is not constant and, of course, depends on the extent of the disease. There may also be slight limitation of motion on the affected side.

On palpation and percussion the tactile fremitus is not often obviously increased, but there is commonly a slight comparative dullness, often best noticed in the supra-spinous or supra-clavicular fossæ.

On auscultation, when the respiration is natural, the inspiratory sound is often diminished as well as the expiratory, which is, however, usually prolonged. When the respiration is increased in vigor, the inspiration may appear like the other side, though sometimes harsher, but the prolongation and higher pitch of the expiratory sound becomes much more differentiated from the other apex. A good place to hear this is often in the triangle between the scapula, the spine and the curve of the neck. The voice sounds are louder and nearer the ear, particularly with whispers. Râles are usually absent, unless the patient has recently "caught cold."

I consider, then, that the combination of slight dullness, prolonged expiratory murmur, and increased vocal resonance at either apex, particularly the right, renders a case suspicious, and if these signs be combined with low weight, constantly rapid pulse, or a family history of tuberculosis, I treat the case as tubercular, even though bacilli and elastic fibres are not found. The sputum has often been negative in these cases, as it often is even in known tubercular cases, though possibly better methods may be able in future to find the bacilli in these cases, or blood examination throw more light upon the diagnosis.

I know that these three cardinal signs are not generally accepted as sufficient to make a diagnosis upon, and are referred to a normal variation of the right over the left apex.

My reasons for a positive belief in their significance are as follows:

- (1) The great frequency of pulmonary tuberculosis.
- (2) I have demonstrated in surgical cases among men with good history and fine physique the absolute similarity in these signs of both apices.
- (3) I have demonstrated by the x-ray the presence of solidification in these cases.
- (4) I almost always find these signs in cases that have tuberculous lesions elsewhere in the body.
- (5) I have watched some of these cases go on to full development of the disease in a well-recognized form.
- (6) These signs sometimes appear on the left side

when they could not be due to a right-sided variation.

In children the diagnosis of pulmonary tuberculosis is, I believe, difficult, but I am always suspicious of any unilateral trouble that is not distinctly lobar pneumonia.

Dr. Morrill, in "Keating," says "that broncho pneumonia is often the starting-point (?) of tuberculosis in children."

The evident conclusion from what has been said is in favor of a greater frequency of tuberculosis than is commonly believed, and this coincides with the statistics at the beginning of this paper. It is worth while to note that the prognosis of many cases as regards the ability to maintain life is good, and those individuals who have a natural expectancy of long life will *ceteris paribus* combat the disease most successfully.

I wish, in closing, to say a few words about treatment. My experience with tuberculin and anti-tubercle serum has led me to the conclusion that neither has a specific action on the disease, and, although Von Ruck and Trudeau report better results from tuberculin than from other methods, I cannot help feeling that it is Asheville and Saranac rather than tuberculin that is responsible. If tuberculin or a serum is superior, it must demonstrate its superiority in every city and town. It seems to me fallacious to assume that a disease with the chronicity of tuberculosis is going to react like an acute self-limited disease such as diphtheria. I believe that our resources are still to be found in sunlight and fresh air, rest and hypernutrition. My rule is, recumbency in the open air every fair day (following definition of weather bureau) for seven hours. This may be maintained, even in winter months, if the body temperature be kept up. If under such conditions a patient can gain, the question of a change of climate is open, otherwise I believe any change will be futile.

The question of nutrition is too large to consider here, but it means oftentimes all the resources at the physician's command.

THE HOMICIDE OF MRS. HATTIE E. MCCLLOUD.¹

REPORTED BY F. J. CANEDY, M.D., SHELburne FALLS, MASS.

THE ninth day of January, 1897, was like one of the rare and balmy days of October; and as the sun rose from behind the eastern range of hills at Shelburne Falls and flooded the valley of the Deersfield with light and warmth, the somewhat sharp frost of the previous night was soon dissipated; and so clear was the sky and soft the air that there was nothing to remind us that we were at midwinter, unless it was the leafless branches of the trees and the brown of the landscape.

Most of the twenty-five hundred inhabitants of the pleasant village of Shelburne Falls, so picturesquely located among the hills on both sides of the Deersfield river, had been for some hours at work in factory or store, or about their business of the streets, when a messenger came hurrying down from the hillside above the town for the medical examiner, with the information that Mrs. Hattie E. McCloud, an educated and refined lady, daughter of County Commissioner Geo.

D. Crittenden, was lying dead in the woods a half-mile out of the village.

It was at about half-past nine in the forenoon that I entered the wooded slope where the body lay surrounded by some twelve or fifteen men, among whom was the father of the dead woman. Her father, coming forward to meet me, stated that his daughter had left home on the previous afternoon at about four o'clock to do some errands in the village, and when she did not return in the early evening, as expected, it was supposed by her family that for some cause she had decided to stay over night with her married sister living in town, and that for this reason no great anxiety was felt for her absence and no search made. He also said that she was subject to fainting turns, and had probably slid down into this place in one of these spells and died from exposure to the cold, and that he didn't think there had been foul play in the case.

Mrs. McCloud was lying on her back seventy feet below the road on sloping ground with her feet down the hill, the point where she lay being connected by a path with the road leading from the village to her home. She was dressed in the usual winter clothes worn by a lady in her position, the outside garment being a loose cape with a fur collar fastened at the neck by a button. There was little disarrangement of her clothing; and her legs were extended, somewhat separated, and covered with her skirts to the knees. Observing the livid color of her face, with froth about the lips, I unbuttoned and turned down the collar of the cape, which revealed discoloration marks with skin abrasions over the larynx and upper portion of the trachea. On raising the skirts I found she had on open knit drawers, which were blood-stained about the genitals. Calling the attention of Mr. Crittenden to the marks upon her throat, I told him there was reason to suspect that her death had been by violence, and that the body and all circumstances connected with her death would have to be subjected to a careful investigation. After noting as carefully as possible the position and condition of the body as found, and ground and objects about, I had it placed on a horse blanket brought from my carriage, and carried by the hands of six men an eighth of a mile up the hill to her home. Before leaving the locality I requested the people to leave the ground, and stationed a man near by in the road to see that no one went upon the spot. I then followed the body to the house, had it laid upon a lounge in a cold room, with none of the clothing disturbed, and returned to the village to telephone District-Attorney Hammond, at Northampton, for an order for an autopsy and an officer. At 11.30 State Detective Benson Munyan arrived, bringing the order for the autopsy, which, assisted by Dr. F. H. Zabriskie, of Greenfield, I proceeded to make at half-past one o'clock in the afternoon.

The description of the autopsy is best given in my report to District-Attorney Hammond, which I will read from this point:

"To the District Attorney of the Franklin District: You are hereby notified that on the ninth day of January, in the year one thousand eight hundred and ninety-seven, at nine o'clock A. M., I received notice that there had been found lying within the said district the dead body of a woman, who was supposed to have come to her death by violence; and upon receiving said notice, I forthwith took charge of said dead body, which I found lying on its back in the woods

¹ Read before the Massachusetts Medico-Legal Society, October 6, 1897.